Johnson (W. B)

Amblyopia from Suppression of the Visual Image.

—BY—

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Reprint from The Ophthalmic Record, November, 1893.

NASHVILLE, TENN.:
A. R. GRAY & SON, BOOK AND JOB PRINTERS
1893.

AMBLYOPIA FROM SUPPRESSION OF THE VISUAL IMAGE.*

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In convergent strabismus, except the squint be of the alternating variety, there is present an amblyopic condition affecting the squinting eye, in which there is more or less marked diminution of the vision.

Hypermetropia was thought by Donders and many other writers to be the most frequent cause of convergent squint, constantly exciting excessive efforts at accommodation, resulting in convergence, confusion of images and subsequent mental suppression of the visual image of one eye. finally terminating in an amblyopic condition, in which the ability to see varied, vision being more acute in some cases than in others.

It has also been claimed by Schweigger, Alfred Græfe and others that amblyopia is not caused by squint, but exists as a primary or congenital condition probably caused by some change, structural or otherwise, in the eye itself or in the nerve

* Read at the twenty-ninth Annual Meeting of the American Ophthalmological Society at New London, Conn.

centers. This produces convergence in consequence of confusion of images caused by deficient sight.

The variety of amblyopia or suppression of the visual image in question is apparently a purely physiological condition. Ophthalmoscopic examination indicates a perfectly normal fundus, although the eye is generally hypermetropic. The nerve centers must certainly be affected by the continued mental suppression of the visual image and their functions finally practically suspended. Unless an operation be performed, which results in re-establishing binocular fixation and fusion of the retinal images, the amblyopia persists, being a progressive mental condition made permanent in the nerve centers by their loss of use and exercise of the power of vision. Amblyopia may be and sometimes is overcome, when it is first established, by a proper correction of visual defects; the commencing squint and the increasing loss of sight may thus be entirely prevented without resort to operative interference. Squint generally appears at an early age, when satisfactory examination is not practical; the amblyopic condition may be present but not demonstrable; for that reason the time of loss of vision can not be definitely ascertained and the question of the probable presence of amblyopia before the onset of the squint, or of its onset as a result of the squint, is very difficult of solution and proof. It is presumed that an amblyopia may come on as a result of squint and persist forever after even though the eyes are brought to a state of apparently perfect parallelism by operation. The vision in the amblyopic eye rarely, if ever, becomes equal to the vision of the fellow eye. The squinting eye cannot, does not, and will not enter into the visual act and has no ability to, and can take no cognizance of any object which appears on its visual axis, the power of suppressing images having become a condition and the vision decreased to such an extent that the eye is practically sightless.

The case here reported, which presents the character and history of many other cases of concomitant convergent strabismus, shows conclusively that, whatever the change which led to the loss of vision, it was not structural either in the eye ball or the nerve centers, but was in all probability a pure case of amblyopia which resulted from the long continued mental visual suppression induced by the confusion of images caused by the loss of parallelism of the eyes, and that the amblyopia was in all probability the result of the squint.

The amblyopia having entirely disappeared after the loss of the fixing eye, when all the existing conditions were changed, indicates the certainty that in this case amblyopia was a condition and not a disease The remaining eye which had been apparently almost sightless, having become excessively amblyopic, after instruction and exercise designed to assist the visual effort, gradually increased its power of vision until perfect sight resulted and the sensitive point returned to the region of the macula lutea. The results of any passed amblyopic condition entirely disappeared, the eye-ball itself and the nerve centers returning to a perfect state of health and visual acuity.

T. McK , age 19, June 3, 1887. File forger. Has been cross-eyed since he was three years of age, and states that during his recollection he had been unable with the left eye to discern any object and define its character.

He has a manifest hypermetropia and constantly fixes with the right eye.

R. V. = $\frac{20}{15}$; $\frac{20}{15}$ w. plus 1.25 D.

L. V. = fingers at 6″, no improvement with glasses.

The fundus was apparently normal.

He applied for treatment intending to have his squint corrected by tenotomy.

June 13. While working at forging, a hot file flew from his tongs and struck him in the right eye.

Two hours after the injury the eye-ball was examined, a large wound of the globe was discovered, having extremely ragged edges and involving almost the entire globe, cutting through the cornea, iris, lens and sclerotic in the cilliary region. Enucleation was advised and performed in the usual manner in the afternoon of the same day.

June 18. The patient was doing nicely and stated that he believed he could see better.

L. E., V. = fingers at 3′

Ophthalmoscopic examination discloses a perfectly normal fundus and a hypermetropia of plus 1.50 D.

June 19. He was first instructed in locating letters on the test card. His field of vision was limited to any object upon which his attention was fixed. If placed directly in front of a test card with the region of the macula in the axis of vision he could see $\frac{3}{200}$. If allowed to read the letters on a plane of his own choosing, bringing the hypersensitive retinal spot into use, and wearing plus 2. D. the test card would appear to be thirteen inches to the left of its actual place of hanging, but he was able to read $\frac{2}{70}$ and as his instruction was continued he read $\frac{2}{18}$, and at times $\frac{2}{12}$ and $\frac{2}{8}$, the letters being moved ten inches to the left of their actual position. He finally read $\frac{4}{15}$.

June 20. After fifteen minutes instruction he was able to read $\frac{20}{30}$ w. plus 1·25 D. on a new test card, never seen before, stating that in order to see the card he was obliged to look to the left of it, although he apparently saw it directly in front of him. He could read $\frac{20}{200}$ without a correcting glass. Although there are six cards on the test frame, he insisted that he could only see one of them at a time and that in its false position.

June 21 to 25. He has been instructed daily with constant improvement in the field of vision and in the rapidity with which he could locate the letters and cards, plus 1.75 D. having been ordered and worn constantly.

June 26. He is able to select letters on any of the test cards and now locates the card in its exact position, and can see all six cards at once without special effort. His vision for near was tested for the first time since the loss of his eye with plus 1.75 D. He was able to read Jæger No. 7, but in locating a word on the test paper, with a pointer, he would point considerably to the left of its actual position.

July 1. The improvement has continued daily, since last date. He can read $\frac{20}{15}$ with his correcting glass, and Jæger No. 1, at 12 inches, locating the words with a pointer in their correct places. He dsclined to accept the plus 1.75 D. he had been wearing and was ordered plus 1.25 D His visual field is normal.

Jan. 23, 1890, nearly three years after the loss of his eye.

His vision $= \frac{20}{15}$ with or without plus 1.25 D. He reads Jæger No. 1 with or without any correcting glasses, although he prefers his plus 1.25 D. for reading.

He never has had any pain, or discomfort, or any blurring of his sight since last examined, and has worked at his trade constantly since that time.

DISCUSSION.

Dr. Samuel Theobald, Baltimore:

The case is an exceedingly interesting one and I am not aware of a similar one having been reported, where through the loss of the good eye, the amblyopic squinting eye being forced to take part in vision, has so rapidly and completely recovered visual power. I think that the Doctor is justified in the view that he has taken of the significance of the case. It is extremely improbable that a *congenital* amblyopia would entirely disappear as happened in this instance. The case offers strong testimony I think in favor of the view combated of late years by Schweigger and Alfred Græfe, but which I attempted to defend several years since*, that the amblyopia of squinting eyes is a consequence and not a cause of the squint.

* Trans. Am. Ophthal. Soc., 1888.

Dr. Herman Knapp, New York:

This case is the best evidence of amblyopia ex-anopsia that I know of. I have had one case somewhat parallel. An old lady had cataract in an eye that had squinted from youth. She was operated on by Schweigger and I had to make an operation for secondary cataract, by which she got vision $\frac{20}{XXX}$. This case comes near to that of Dr. Johnson, but it does not furnish a complete evidence of amblyopia ex-anopsia and subsequent improvement of sight as the latter had not been tested before the cataract developed.

Dr. Henry D. Noyes, New York:

This is the first case in which I feel any confidence in the assumption that the amblyopia was acquired. It is the only one I know of, equally remarkable, except one of Javal. Javal had a case in which he brought the vision up to normal after three years exercise. It must nevertheless be distinctly stated that a case like this only proves that very defective sight belonging to the unused eye, is capable sometimes of remarkable improvement. It does not prove anything as to the source of the defective function. The two things are logically entirely distinct from one another. I am willing to concede that, in a portion of cases of strabismus, the amblyopia may be acquired and is not congenital, but I am far from being convinced that the majority of them are of that character. I have examined an enormous number of cases in which there is congenital amblyopia of one eye without squint. I intend to put on record my cases of congenital monocular amblyopia with refractive error. These cases are much more common than is usually supposed.

In regard to this particular case, I should have been glad if examination had been made to determine whether or not there had been color scotoma at some period of the history. Color scotoma is very common in amblyopia and strabismus. Further-

more, other conditions obtain. I recently saw three cases of strabismus. In one, the squinting eye had an absolute scotoma of ten degrees, with absolutely no light perception. The patient was a woman 28 years of age who had been operated on in each eye, but the squint had not been cured.

The most probable explanation in favor of the view that squint amblyopia is acquired, is on the assumption that the cerebral part of the visual function is in suspense, and by continued suspension loses conscious activity. This however is a very different thing from the usual assumption which makes the lesion ocular.

In opposition to this view are numerous cases where persistent scotoma, persistent dimness of sight argues in favor of a local lesion in the eye, which I do not believe is the result of disease.

An interesting feature that Dr. Johnson records is the improved projection in looking at an object. It seems to me that might have been corrected by a tenotomy of the internal rectus. In time, he overcomes that. That is a fact that is frequently observed in dealing with strabismus when you attempt to restore binocular vision.

I may perhaps make this remark with the view of assisting in the determination of central scotoma: If you have a patient whose vision in the amblyopic eye may be $\frac{3}{x}$ and type consisting of a line of five letters; you ask him to look at the middle letter and he will tell you that he can not see it, but that he sees those on either side. Then you may change his point of fixation from one point in the line to another. You will find that the object on which he attempts to fix is not clear, while objects excentrically placed are seen. This is better than the perimeter. You can do it with the perimeter or better with the black-board.

Dr. Samuel B. Risley, Philadelphia:

I think that the existence of amblyopia ex-anopsia in con-

vergent strabismus can no longer be doubted. I some time ago recorded a group of such cases in which the presence of amblyopia from disuse was demonstrated. In one case the right eye which was amblyopic had been operated on to correct a strong convergence and after four years the case came under my observation, the left eye now being the squinting eye. The right eye before the operation had been almost blind, but was found with V. = $\frac{6}{7}$. In each eye there was hypermetropia of 3 D. She could barely count fingers with the left, fixation being eccentric. The right eye had normal acuity of vision, $\frac{6}{6}$. I did tenotomy on both interni, corrected the refraction and insisted upon exercise of the amblyopic eye for half an hour, morning and evening, the right eye being excluded by a bandage. Vision steadily improved so that at the end of three months it had come up to $\frac{6}{7}$. Here was an instance where the amblyopia had existed at different times in each eye and had been recovered from.

Another case was that of a small boy aged 2 or 3 years who was brought to me with developing convergent strabismus. I followed my usual habit and placed the eyes under a mydriatic, gave smoked glasses and exercised the right eye, which showed the greatest tendency to convergence, and followed the boy through three years. Finally he had learned his letters and I found that he had vision of $\frac{6}{7}$ or $\frac{6}{8}$ in each eye with correcting glasses. He had hypermetropic astigmatism with asymetrical axes. Finally after an absence of six months, he came back and vision had sunk to $\frac{1}{3}$ in the right eye and maintained its former standard in the fellow eye. I then placed an opaque disc between the glass and the eye on the right side, and had him wear this morning and evening. Under this, vision came up once more to $\frac{6}{12}$ and some letters in $\frac{6}{9}$. In other words, it distinctly improved under exercise, and the exclusion from vision of the fixing eye. After watching the case for a few

weeks longer, I did tenotomy on the right eye with the result of correcting the tendency to convergence and restoring binocular sight. Two weeks from the date of the operation, vision was $\frac{6}{6}$ in each eye with correcting glasses. I think that with the evidence of such experience, we can no longer deny the existence of amblyopia from disuse, and must admit that all these cases certainly are not congenital, since the amblyopia developed under observation and disappeared under the measures adopted for its relief.

Another case which has already been published was that of a child, the daughter of a physician, who had convergent strabismus with vision reduced to one-half in the squinting eye. I corrected the existing hypermetropic astigmatism with the result that the strabismus, which before had been fixed, became periodical. She came back after several years, on account of headache following work at a near point. The strabismus had been so entirely removed by the use of glasses, that I did not suspect it to be a case of strabismus until the former record was consulted. Vision was perfectly normal in each eye, i. e., the amblyopia had disappeared. In view of these clinical facts it is futile to deny the occurrence of amblyopia from disuse.

Dr. B. Alexander Randall, Philadelphia:

Cases of cure of amblyopia ex-anopsia will but rarely be recorded in spite of the challenge of these who deny such a condition.

These cases must always be difficult to definitely settle, because of the youth of many of the patients and the difficulty of applying the tests of vision in such cases. A case that has greatly interested me is one that has grown up under my eye. Strabismus occurred periodically in the second or third year, always with fixation of the right eye. Mydriatics relieved the habit to a certain extent, up to about the fifth year; but the strabismus then became nearly fixed and glasses seemed neces-

sary. A glass of some 6 D. was given and under its use the child began to see, she could learn her letters and I was able to determine that the now slightly converging eye had $\frac{20}{xx}$ contrasted with $\frac{20}{xxx}$ in the right. To-day, the hypermetropia remaining the same, and the case having never been operated upon, vision is $\frac{6}{6}$ in each eye, although the tendency to strabismus has never been wholly overcome. She has full normal vision, rather better in the left eye, which still tends to converge; and turns strongly in the moment her glass is removed, and ordinarily she has no difficulty with vision with both eyes. The eyes working in perfect harmony with normal vision.

Each of the other less hypermetropic children has had periodic strabismus; but has escaped with only a need for glasses at near work, vision having always been normal in each eye.

Dr. Samuel Theobald, Baltimore.

The region in which the highest grade of amblyopia exists in the squinting eye has I think an important bearing upon the question, whether the amblyopia is due to the squint or is a congenital condition. The retina is most amblyopic about the yellow spot, and to the nasal side of this region in convergent strabismus; while in divergent strabismus it is the macula and the temporal portion of the retina which are most affected, just where it is most important in order to prevent diplopia and confusion of vision, that the retinal image in the squinting eye should be suppressed. The other portions of the retina are much less amblyopic. A case illustrating this point has occurred to me. A young woman of 25 years had a convergent squint without diplopia. I did a tenotomy upon one internal rectus, leaving a considerable residual squint, and still no diplopia. Then I divided the internal rectus of the other eye, and got a slight over-correction, and at once there was diplopia. The moment the retinal image through the over-correction produced

by the second operation fell upon the outer side of the yellow spot, there was diplopia, the vision in this region of the retina having remained comparatively unaffected, because the images formed upon it, caused little or no confusion of binocular sight and so were not mentally suppressed.

Dr. E. E. Holt, Portland, Maine.

Several years ago (1885), I read a paper before the Society entitled, "Strabismus, its correction when excessive and in high degrees of Amblyopia", in which I described the method I had then practiced seven years of advancing the weakened, attenuated muscle in conjunction with tenotomy of its opponent which maintained the deviation in these cases. I reported a case in which this double operation had been performed when several tests showed the eye operated upon was blind, but after the operation, finding there was sight, practice brought the vision up to $\frac{1}{24}$. Some of the members of the Society doubted the accuracy of the records, although it was related that several tests were made with the same result. I think continued practice with strong lenses will give surprising results in some cases, but it requires patience and perseverance.

Dr. W. B. Johnson, Paterson, N. J., closing:

It seems to me that the peculiarities, in this case, are the length of time that the amblyopia had persisted, the return of perfect vision, and the shortness of time required for the vision to become normal. I was surprised to hear cases reported to-day in which, after operation for squint, vision, in the amblyopic eye became perfectly normal. In my case vision became normal, in the amblyopic eye, but there was no counteracting features on account of the loss of the eye, which had previously performed the entire visual act. In my experience, I have failed after operations for squint to find perfectly normal vision restored to the squinting eye, even when binocular single vision was attained.

The physiological central scotoma which Dr. Noyes speaks of was present. The period of time required for the return to normal vision was, from the thirteenth of one month to the first of the following month, the vision then being $\frac{20}{15}$, with a correcting plus lens and subsequently becoming $\frac{20}{15}$, with or without such correction. It would seem to be established, that almost complete loss of vision from amblyopia certainly can and does exist without any diseased condition being present either in the eye itself, or in the nerve centers; that amblyopia is in that case due to the continued "supression of the visual image," which is a condition self-limiting under certain circumstances, and not a disease either causing or caused by stribismus.